VISUAL DISTURBANCES IN TABES DORSALIS, AND AN ATTEMPT AT A COMPREHENSIVE EXPLANATION OF THE SYMPTOM-COMPLEX OF TABES.

Dr. E. Berger's remarkable paper upon this subject appears in English dress in the "Archives of Ophthalmology" (October, 1890), translated by Dr. Ward A. Holden. The symptoms of tabes, besides those caused by disease of the spinal cord, consist of disturbed function of special cerebral nerves, sometimes of the cerebrum and probably of the cerebellum.

Of the general disturbances of the brain should be mentioned headache, disturbance of speech, failure of memory, and disturbances of intellect; yet these are unusual. More frequently cerebral nerves are affected—the optic nerve, the nerves of the ocular muscles, and single branches of the fifth. It is seldom that the hypoglossal, the glossopharyngeal and the olfactory nerves are also affected. Occurrences of diabetes mellitus and insipidus, of bronchial asthma, of nephralgic crises, and of rectal and urethral colic are reported.

Pathological changes are found in the medulla, in peripheral, cerebral and spinal nerves, in the spinal ganglia, and in the sympathetic nerve. Disease of the optic nerve appears first in the periphery, and affects the central fibres later. The nerve fibre and ganglionic cell layers become atrophic, owing to increase of connective tissue, that begins with a formation of fat drops and an increase of pigment (Leber, Raymond, Arthaud). The author thinks that his own experiments on animals would confirm the idea that disease of the medulla can explain the symptoms of tabes, as here may be found the connecting link between the spinal and the optic nerve affection.

The analysis of one hundred and nine cases shows that tabes appears most frequently during the most vigorous period of life—between the ages of thirty and forty-five years. It has appeared as early as the age of fifteen, and as late as seventy. The cases occurring at the earliest and latest periods of life are free from severe complications. These occur during the period that is normally of greatest activity. Of the cases studied, there were forty-four men with syphilitic history and forty-nine non-syphilitic; among the women, three syphilitic and thirteen free from specific taint. On an average, the first symptoms of tabes appeared eleven and a half years after the primary affection of syphilis.

Tabes in syphilitic cases begins for the most part with cerebral symptoms, while in the non-syphilitic the spinal symptoms manifest themselves earliest.

There is often a slight narrowing of the palpebral fissure, even when no paralysis of the oculo-motor nerve exists, and epiphora is about half the cases, together with fibrillary twitching on closing the lids, and a diminished intra-ocular tension to the extent of causing the ball to be quite soft. Inequality of the pupil often exists. An irregularity, however, hitherto unnoticed, is in the shape of the pupil. It is often elliptical, with its long diameter most frequently oblique from downward and outward to upward and inward, though the long diameter may lie horizontally or in some other direction. Their action to light is lost first; later the reaction to accommodation. This peculiarity is found also in progressive paralysis; otherwise it is rare. Stolzenberg found it in many cases of cerebral syphilis. Bisulphide-of-carbon poisoning has produced it (Uhthoff). Mauthner's explanation of pupillary immobility, verified by the experiments of Moeli and Knapp, is, that the fibres for the pupillary light reflex, lying in the wall of the third ventricle, suffer from a chronic ependymitis and a consequent sclerosis.

Cases with paralysis of ocular muscles are more disposed to optic atrophy than those in whom this paralysis is absent. The left eye is first affected twice as often as the right. The greater number of atrophies develop in the *stad. præatact.* If this stage is once passed, the danger of its occurrence is less. Should an optic atrophy develop later, the consequent blindness follows more rapidly.

Erb gives the following as the usual order for the loss of color-perceptions: (1) green, (2) red, (3) yellow, (4) blue. No relation exists between the contraction of the visual field, the color-fields, the disturbance of central vision, and of color-perception.

With regard to the blood-vessels of the eye, the capillaries must be dilated, at least at the beginning of the atrophy. The central vessels undergo the following changes: (1) arteries and veins normal; (2) arteries narrow, veins normal; (3) arteries narrow, veins dilated; (4) arteries and veins narrow. The two groups of functional visual disturbances, apart from dioptric errors, are: (1) those in which the fault lies in nerve-fibre conduction, and (2) those in which the functional error is caused by a disturbance in nutrition. The causes of the visual disturbances must be sought for in the vessels alone.

Many facts point toward vaso-motor disturbance from central causes. In regard to the treatment of tabetic optic atrophy, mercury is positively harmful. If injections of strychnine do no good, rest in a darkened room and iodide of potassium are in order. Among syphilitic patients there is greater tendency to permanent and multiple paralyses of ocular muscles than among non-syphilitic. Paralysis in the regions supplied by the facial nerve also occurs.

Thus it will be seen that many of the symptoms of tabes may be explained by a lesion of the nerve-nuclei in the fossa rhomboidalis and its continuation into the aqueduct of Sylvius. The difficulty in closing the lids is probably due to a slight functional disturbance of the upper nucleus of the facial nerve, which is in relation with the nucleus of the fifth nerve; the myosis, to paralysis of vaso-constrictors in the medulla; the reflex iridoplegia, to a lesion of the fibres running in the wall of the third ventricle to the nucleus of the sphincter pupillæ; and the diminished intra-ocular tension—existing in one-third of the cases—to lowering of the tone of the vessels. Lachrymation in tabetic patients is a vaso-motor neurosis, analogous to the unilateral hyperidosis that is sometimes present. The sympathetic nerve is not the cause of certain ocular symptoms, but the medium by which these are carried from the central nervous system to the eye. A number of nerves whose nuclei lie in the medulla may become affected. The whole facial nerve, or only its lower twigs, may become paralyzed. The auditory, the vagus, the fifth, and rarely the spinal accessory may suffer functionally, and, according to some authorities, the glosso-pharyngeal and hypoglossal nerves as well. In general, the nerve-nuclei in the medulla nearest the middle line suffer more frequently than those further outward, and the nuclei in the upper portion more frequently than those lower down. An affection of the medulla is indicated by the development of diabetes mellitus in tabes, and by febrile symptoms. The author then states that the following points are to be considered:

1. Whether we are justified in assuming the vascular centres to be located in the medulla.
2. That, in consequence of disease of the nerve centres, vascular changes can develop.
3. That vascular changes occur in the various nerve regions which are the seat of the morbid process in tabes.
4. That the vascular changes are also the cause of the disease of the nerve-tissue.

5. Whether trophic nerves cannot cause simultaneously the changes in the affected nerve regions.

A careful survey of these important considerations seem to the author to support his view of tabes dorsalis.

THE EFFECT UPON THE BODILY TEMPERATURE OF LESIONS OF THE CORPUS STRIATUM AND OPTIC THALAMUS.

W. Hale White, in the January number of the "Journal of Physiology," has a paper with this title. Hitherto, in experiments upon animals, but little attention has been paid to the normal temperature of the animals operated upon, and but little to the effect of the anæsthetic or the trephining apart from the injury. The author's experiments lead him to believe that the corpus striatum and optic thalamus have, in rabbits at least, the power of modifying the temperature of the body, and that the surrounding white matter has no such power.

KNEE-JERK AND ITS PHYSIOLOGICAL MODIFICATIONS.

The "Journal of Physiology," January, 1890, contains a series of interesting experiments that demonstrated the changes in knee-jerk from purely physiological causes. Exaggerated reflex can be inhibited by the stimulation of the skin or peripheral nerves (Nothnagel, Lewinski, Erb.); but the reflexes thus inhibited were in nearly all the cases reported the clonic movements produced under morbid conditions, by stretching the tendons of the muscles concerned. Erb mentions a case of myelitis from compression in which tendon reflex and trembling of the legs was stopped at once by pinching the skin of the abdomen. If the patient clinches the hand when the patellar tendon is struck, or makes other violent movement, the coincident jerk is increased (Jendrássik). Volitional acts directed to other parts of the body, painful stimulation of the nerves of the skin by pinching, by the application of heat, cold, or electricity, exposing the eyes to the light of burning magnesium wire, will increase knee-jerk (Mitchell and Lewis). Fatigue, hunger, enervating weather, sleep—conditions which decrease the whole activity of the whole central nervous system—decrease the average patellar tendon reflex. Rest, nourishment, invigorating weather, and wakefulness increase it (Lombard).